THEORETICAL ESSAYS

Sleep Science

Apnea-hypopnea index in sleep studies and the risk of over-simplification

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ABSTRACT

According to recent reports, sleep disorders affect 30% of the adult population and 5-10% of children. Obstructive Sleep Apnea Hypopnea Syndrome (OSA) has a considerable epidemiological impact and demand for consultation is growing in our community. Therefore, it is necessary to know the principles of interpretation of diagnostic methods. A suspicion of OSA requires confirmation. According to the guidelines of the Argentine Association of Respiratory Medicine, polysomnography (PSG) is the gold standard for OSA diagnosis, while home sleep testing (HST) can be accepted as a comparatively effective method depending on the clinical situation of the patient. This article questions the use of AHI (apnea-hypopnea index) as the only measurement needed to diagnose OSA and assess its severity. In fact, it is surprising that, despite the large mass of data analyzed during sleep studies, current practices only focus on AHI. More than four decades have passed since OSA was first described. Our tendency to oversimplify complex conditions may prevent us from gaining a deeper and more thorough understanding of OSA. The development and validation of OSA severity scoring systems based on multiple parameters is still a pending issue.

Keywords: Sleep Apnea Syndromes; Severity of Illness Index; Sleep Disorder.

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INTRODUCTION

According to recent reports, sleep disorders affect 30% of adults[1,2] and 5-10% of children[3]. The current obesity epidemic could increase such percentages[3-5]. In Europe, OSA accounts for 30% of consultations to pulmonologists[6]. In our community consultations due to OSA are also increasing.

According to American Academy of Sleep Medicine guidelines, polysomnography (PSG) is the gold standard for diagnosing OSA, while home sleep testing (HST) can be considered a comparatively effective method (but not an exact equivalent) depending on the clinical situation of the patient[7,8] (i.e. symptoms, discomfort, risk, history of associated conditions). Such context leads us to ask: How must we interpret information should be obtained from sleep studies?

Early PSG findings (in the late '70s) identified pauses of breathing (apnea) based on changes in inhaled/exhaled air temperature and resulting damages: fragmented sleep (electroencephalography) and cardiovascular instability (changes in blood pressure or heart rate), which cause sleepiness[9,10]. The definition of the disease and normal cutoffs are of those time[9].

Apnea Index (AI) became the first indicator to define OSA since obstructive apnea is its most distinctive element[9,10]. Subsequent improvements in devices and methods (flow/pressure cannulas) to measure airway collapse added to the complexity of classifying and quantifying obstruction events[11-15]. Partial collapse (hypopnea) has a similar effect (though it is still unknown to what extent), causing arousals and/or O_2 desaturation. As the effect of partial collapse became recognized, it was included in the definition of OSA creating the AHI index which we now use[11-16].

The description of obstructive events should be, in theory, a simple procedure just by following the guidelines for the interpretation of sleep studies, which are updated as new knowledge becomes available[11-15]. However, the definition of hypopnea remains a major challenge, since there is no consensus over the level of airflow reduction necessary to classify an event as hypopnea[13-16]. Even in this scenario, physicians make an extensive and oversimplified use of AHI assuming the biological effects of apnea and hypopnea are basically the same[16] and analysis of sleep studies focuses on improving AHI accuracy and defining events with demonstrable consequences in artifact-free recordings[16,17].

Some definitions are based only on events associated with significant O_2 desaturation and others try to adjust for underlying variations considering only those respiratory events that cause a physiological response (e.g. micro-arousals)[17]. The use of simplified diagnostic strategies has entailed the description of arousal surrogates (movements, change in heart rate or arterial tone) as signs that supplement AHI[16-18]. Ho et al.[17] studied the impact of different definitions of hypopneas (associated with different thresholds of oxygen desaturation and arousal), in a detachment from the original Sleep Heart Health Study in >6000 subjects and they show that three methods of scoring hypopneas yielded significantly different estimates of the apnea-hypopnea index (AHI), although the relative difference is reduced in severe disease.

Since the original description of AHI, a large body of evidence has linked OSA with clinical consequences like; excessive sleepiness, deterioration in quality of life, traffic accidents, diabetes and insulin resistance, hypertension (HT), stroke, heart failure, and mortality[7,15-21]. Almost all studies use AHI as an indicator of exposure to respiratory events during sleep. In addition, intervention studies (CPAP) have shown that OSA treatment is associated with better outcomes when AHI goes down[22].

It is surprising that despite the large mass of data analyzed during sleep studies, OSA severity rely on AHI. Even though AHI is widely used as a predictor of OSA-related complications, its use has several limitations. First, AHI gives us an idea of the frequency of respiratory events during sleep time, but does not allow us to know the magnitude of oxygen desaturation, which may affect other organs and should be included and interpreted in PSG or HST reports[15,21-23]. OSA is a model of intermittent hypoxemia characterized by cycles of hypoxia and re-oxygenation of short duration (15 to 120 seconds) occurring over 6 to 8 hours of sleep for many years. Both animal and human models of chronic intermittent hypoxemia appear to show a significant role in the pathogenesis of OSA comorbidity, including hypertension, cardiovascular events, diabetes, neurocognitive impairments and cancer[24,25].

In order to determine what degree of hypoxemia is associated with increased morbidity and mortality in patients with OSA, it is necessary to establish whether different patterns of oxygen desaturation independently predict the development of cardiovascular events and another outcome of interest. In that sense, several publications have observed that patients with OSA the risk of cardiovascular events, recurrence of atrial fibrillation after successful cardioversion, sudden death, and neurocognitive impairments, were observed in those patients with a greater degree of oxygen desaturation[24-29].

Second, AHI does not consider the duration of apneas/hypopneas. It is not reasonable to assume that a 10 seconds (s) apnea/hypopnea is equivalent to 30 or 60 s event, in terms of hypoxemia or hypercapnia, development of negative intrathoracic pressure, changes in heart rate or blood pressure and arousal reaction. Third, it is also important to note that AHI do not consider the distribution of nocturnal events. Thus, data related to supine/non-supine AHI or AHI in REM/NREM sleep are reported to illustrate the heterogeneity in the distribution of respiratory events[16].

Finally, two OSA patients with a similar AHI may differ in terms of severity depending on their age[30], occupation, daytime symptoms and associated conditions. Likewise, two individuals with the same AHI may present different levels of tolerance and different clinical manifestations[16,23]. New evidence suggests treatment benefits are not the same for patients with a high AHI and no sleepiness[32,33] and other published data highlight the impact of hypoxemia on cardiovascular outcomes[26,34].

It is necessary to develop a score to assess OSA severity and prognosis which, besides AHI and its different variables (total AHI, supine, non-supine, REM/NREM), should include type and duration of respiratory events, O_2 desaturation index (ODI3/4%, SO_2 mean, Time <90%), symptoms (e.g. sleepiness, which sometimes is difficult to measure objectively), body mass index (BMI), and associated comorbidities, since obese and overweight individuals have been reported to have higher mortality rates[35]. Additionally the O_2 saturation behavior may not be the same as that of the respiratory flow when the BMI is increased[23].

Is it possible to consider the following two cases as being equivalent? (1) an individual with OSA with an AHI of 19 events/hour, 34 kg/m^2 BMI, T90 > at 10% of sleep time, daytime sleepiness, and hypertension; and (2) an individual with an AHI of 19 events/hour, 26 kg/m^2 BMI, T90 > at 1% of sleep time, without daytime sleepiness and without hypertension. The answer seems obvious. In terms of AHI, both are moderate OSA cases. However, the first one seems more severe (higher BMI, more hypoxemia, and more risk of pulmonary hypertension)[36]. Our challenge for the future is to stratify risk and prognosis using sleep studies, BMI, and clinical examination.

Night-to-night AHI variability (a phenomenon identified three decades ago) may result in one patient having a normal PSG one night and mild-to-moderate OSA on a different night[37]. These changes could derive from sleep position, changes in the pharynx, and changes in each night's REM/NREM ratio. Biological parameters (e.g. changes in nasal resistance, medication, alcohol and drug abuse) may contribute to this variability. However, the pragmatic application of this information is not fully understood yet.

Another source of error to be considered is inter-observer variability in the identification of hypopnea events. It is estimated that 10% of patients evaluated using PSG could fall into the false negative category for OSA. There is also evidence that the respiratory phenomenon is dynamic, and that there are patients who present central phenotypes that after acute episodes change to obstructive or vice versa[35-38].

Also, since the estimation of AHI by HST is based on total recording time, rather than total sleep time, AHI is usually 15% lower than PSG AHI, which may result in an underestimation of severity[15,16]. In this context, oximetry indicators (O_2 desaturation/hour, time <90%) become especially important[23]. Thus, physicians´ decisions may vary depending on PSG or HST values. A European multicenter study that assessed indication of CPAP based on PSG versus HST (respiratory polygraphy) findings in patients at risk for OSA showed remarkable consistency for >20/h AHIs, but a 20% inconsistency for <15/h AHIs[39].

In summary, though AHI has been extensively used for OSA diagnosis, it entails many limitations when it comes to assessing severity. A high AHI can identify the affected population but intermediate-risk groups are usually left at the mercy of clinicians' management skills. The development and validation of OSA severity scoring systems based on multiple parameters is still a pending issue.

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